and consulting colleagues in sports medicine and neurology including physicians to the British Boxing Federation, I have found no reference to a reversible syndrome of this type following trauma to the eye. Maybe such occurrences have gone unreported because of their transience.

WHERE WAS THE LESION?

The semeiological paradox in this patient was the association of *total* ptosis with *marked* miosis—a combination excluding both Horner's syndrome (where the ptosis is classically stated to be only partial) and a third nerve palsy (where the pupil is usually dilated).

One could speculate that since in this patient there was no history of trauma to the right side of the neck, the bruises over the right eyelid and cheek represented the most likely site of the causal injury. The fact that there had been no symptoms either in the neck or in the right arm made it unlikely that there had been traumatic carotid dissection or damage to the cervical cord or nerve roots.

What about a dual pathogenesis? Could direct trauma to the eye have induced severe (but transient) iris spasm, and trauma to the third nerve in the orbit have caused severe ptosis, without other evidence of ophthalmoplegia? Although this might be feasible, the parallel temporal evolution of the two features would result in the raising of many an eyebrow. The same response would doubtless be triggered if one were to postulate direct trauma to the tarsal muscles as a cause for the ptosis, associated with intraorbital sympathetic injury to account for the miosis.

Finally, could an unusual anatomical substratum account for what was observed? Might this be a variant of Horner's syndrome after all, but occurring in an individual in whom the sympathetic played a greater role than usual in the innervation of the upper lid? Could a sympathetic lesion, in an individual so 'wired', cause both a total (instead of a partial) ptosis as well as the expected miosis? Or is this one heresy too far?

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Tropical ulcers and diphtheria

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Corynebacterium diphtheriae is a well recognized cause of indolent skin ulcers in people who have been in the tropics. In temperate climates, too, it may be cultured from ulcers in socially disadvantaged people such as homeless alcoholics. Such lesions are a more efficient source of respiratory infection than pharyngeal colonization because they contaminate the inanimate environment and bacterial shedding lasts longer[1,2]. In skin lesions the organism can be confused with skin flora such as diphtheroids, and most ulcers also yield an associated pathogen such as Lancefield group A streptococcus (*Streptococcus pyogenes*) and/or *Staphylococcus aureus*[3], which is then presumed to be causal. We report a case.

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CASE HISTORY

A 27-year-old man walked into the Hospital for Tropical Diseases, London on 22 November 1996 with an ulcer on his left hand. He had been in Nepal and Thailand from 25 October to 16 November 1996 and the ulcer had appeared on 2 November after an insect bite. It gradually increased in diameter and needed frequent dressing by his travelling companions. He felt feverish but had no other symptoms. His immunization history included diphtheria toxoid in childhood but not subsequently. On physical examination there was a necrotic sloughy ulcer 4 cm in diameter on the dorsum of his left hand. A swab was taken and he was treated as an outpatient with amoxycillin and flucloxacillin. The swab yielded *C. diphtheriae* susceptible to penicillin and erythromycin. On the day of preliminary identification the organism was sent to the Diphtheria Reference Unit at the Central Public Health Laboratory (Colindale) for further characterization and tests for toxigenicity and the consultant for communicable disease control was informed. Twenty-four hours later the Reference Unit reported toxigenic *C. diphtheriae* var intermedius. The same day the patient was recalled into hospital, put into source isolation, and treated with erythromycin 500 mg 12-hourly for two weeks.

Antitoxin was not given because he had already received 5 days' treatment with two penicillins, he showed no signs of toxicity and the lesion was healing rapidly. Repeat swab from the ulcer on admission to hospital still grew *C. diphtheriae* and a throat swab was negative. During the subsequent week all the patient's close contacts including household and travelling companions were traced and given chemoprophylaxis with oral erythromycin.

COMMENT

Travel history is always taken carefully at the Hospital for Tropical Diseases to assess risks, and in our microbiology department all ulcers from patients with a relevant travel history are cultured for *C. diphtheriae*[4]. This combined approach, together with prompt toxigenicity tests performed by the Diphtheria Reference Unit, direct communication with the consultant for communicable disease control and speedy contact tracing may have prevented the spread of the microorganism in the community.

Acknowledgment We thank the staff at the Diphtheria Reference Unit at the Central Public Health Laboratory (Colindale) for confirming the identification of *C. diphtheriae* and testing it for toxin production.

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Multiple primary haemangiomas of bone mimicking vertebral metastases

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Haemangiomas are slowly growing benign hamartomas which in bones are most commonly found in the calvarium and vertebral bodies. Within the spinal column, the thoracic vertebrae are most frequently involved[1]. Their occurrence at other skeletal sites is rare. Rarer still are haemangiomas of bone involving multiple skeletal sites[2]. Usually it is a solitary incidental radiographic finding of no clinical significance[3], but occasionally haemangiomas of vertebrae cause a syndrome of extradural spinal cord compression[4–7]. We report a case of multiple primary bony haemangiomas mimicking metastatic bone disease in a patient with spastic paraparesis.

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CASE HISTORY

A man aged 45 reported coldness, numbness and clumsiness of the legs. The symptoms had begun ten months before and had become much worse in the past six weeks. They were exacerbated by neck flexion or use of the hands, which caused electric shocks or pins and needles to go down the backs of his legs into the soles of his feet. He was also aware of an upper thoracic burning sensation in his spine without radiation around his chest. He was otherwise fit and well with no significant medical history other than an appendicectomy and a spontaneous pneumothorax several years previously. His mother had a cerebral metastasis from a medullary carcinoma of the thyroid; his father had angina and benign prostatic disease.

On examination he had no abnormalities of the cranial nerves; cervical and lumbar spine movements were good without limitation and there was no spinal tenderness on palpation or percussion. Power, sensation to pinprick, joint position sense, temperature sensation and reflexes in the upper limbs were normal. In the lower limbs, however, there was increased tone and sustained clonus bilaterally with very brisk reflexes and extensor plantar responses. He had normal power in the lower limbs apart from a proximal weakness of flexion at the hips. Joint position sense was absent in the toes and there was a distinct sensory level to pinprick at the third thoracic level (T3) on the left and T4 on the right.

A thyroid scan, done because of the family history of medullary thyroid carcinoma, showed increased uptake in